S142. NOVEL CONCEPTS OF SOCIAL IMPAIRMENTS IN SCHIZOPHRENIA

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Background: Social deficits are a well-known phenotype of schizophrenia, which strongly influence the clinical progress of patients. A core substrate of these dysfunctions are altered Theory of Mind (ToM) processes which critically shape social interactions and can impress in an exaggerated and atrophied form in the disorder. Although there is a well-established link between clinical outcome measures and ToM deficits, less evidence exists about the neural and behavioral mechanisms underlying a specific core function responsible for forming interpersonal mental representations, which in turn aid to optimize social interactions. Along this line, an urgent clinical issue is how alterations in these interpersonal predictive processes translate into clinical issues and whether they can be positively influenced by psychological group interventions.
Methods: In a set of different studies, we used functional and structural magnetic resonance imaging and a dynamic social interactive task, a modified prisoner's dilemma game, in which the participants can form mental representations of different interaction partners in order to optimize their joint interaction sequences. Methodologically, we have drawn on several sophisticated methods ranging from graph theoretic network indices, functional and effective connectivity to structural covariance analyses and linked them with behavioral and clinical outcome measures.
Results: Our data shows a central relevance of the right temporo-parietal junction (rTPJ) in forming mental representations in healthy subjects, given the region integrates memory processing streams during social interactions. Further behavioral analyses indicate that these mechanisms are relevant for interpersonal adaptive processes during social interactions. In patients with schizophrenia, we have found dysfunctions in this important mechanism, indicated by a reduced integration of neural pathways from the temporal lobe, such as the hippocampus. These alterations are associated with behavioral indices of dysfunctions in generating mental representations during an interpersonal interaction. When we examined neural computations in the entire ToM network, we found for the first time that the rTPJ has reduced out-going effective connections to brain regions linked with higher-order cognition, such as the dmPFC, in patients. On a conceptual level, this finding might be associated with dysfunctional updating processes from brain regions located in the temporal lobe, which will be an ongoing empirical question.
Discussion: Our results indicate a central pathological relevance of the rTPJ for social dysfunctions in schizophrenia. First, the regions seem to be less informed by memory processing streams, relevant to update social information by previous information. Additionally, we have shown that in the core mentalizing network the region integrates to a lesser extent into the dmPFC, which might be associated with our previous findings. Our results show concrete targets for specific interventions to improve the clinical important social-cognitive dysfunctions occurring in the disorder. Hence, we suggest approaches to enhance the functioning of brain mechanisms relevant for human connections that can facilitate the patients' clinical outcome.

S143. NEURAL MECHANISMS OF ROBOT-INDUCED HALLUCINATIONS IN HEALTHY PARTICIPANTS AND SYMPTOMATIC HALLUCINATIONS OF NEUROLOGICAL AND PSYCHIATRIC ORIGIN

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Background: The ability to recognize whether sensory consequences have been self-generated or externally produced is an important element of motor control and self-monitoring. Deficits in self-monitoring have been proposed to cause abnormal bodily experiences and psychotic symptoms such as hallucinations. A recent study designed a robotic system that applies sensorimotor stimulation in healthy subjects and safely induces mild presence hallucinations (PH) and passivity experiences (Blanke et al., 2014). PH are defined as the sensation that someone is close by when actually no one is present and passivity experiences are characterized by perceptions or beliefs that an external agent is controlling one's actions, perceptions, and/or thoughts. Although, both symptoms occur in schizophrenia and Parkinson's disease, their neural mechanisms are unknown.
Methods: Here, we first investigated the neural mechanisms of PH and passivity experiences in 25 healthy subjects. We developed a new MR-compatible robotic system able to generate the aforementioned sensorimotor conflicts, while recording subjects' brain activity using fMRI. In addition, we applied lesion network mapping to 11 neurological patients with symptomatic PH and compared both populations to find a common PH network. In a final step, we investigated the relevance of the PH network by analyzing the connectivity of the PH network in resting state fMRI data from 58 psychotic patients.
Results: We first evaluated the regions associated with the general sensorimotor conflict, which revealed the left sensorimotor area, the left putamen, the right inferior parietal lobule and the right cerebellum. Then, we analyzed the regions that were more activated during the condition eliciting PH and passivity experiences and found the inferior frontal gyrus (IFG), the insula, the superior medial gyrus and the middle temporal gyrus (MTG). Comparison of these two networks with the symptomatic PH network in neurological patients highlighted the IFG, the MTG and the vPMc. The resting state analysis within those regions in psychotic patients revealed no global differences between the groups but a functional connectivity decrease between MTG and IFG (bilaterally) specific for psychotic patients experiencing passivity experiences.
Discussion: Collectively, we showed that through the use of a robotic system generating sensorimotor conflicts, the neural correlates of induced-PH and passivity experiences can be studied in healthy subjects in a controlled manner. In addition, we found two networks associated with induced-PH and passivity experience. Of these regions, three were also recruited in patients with PH of neurological origin, forming the PH network. MTG-IFG connectivity in the PH network was altered selectively in psychotic patients with passivity experiences, revealing the relevance of the neural mechanisms of PH and passivity experiences in psychosis.

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